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Case report

Acute mitral insufficiency as a consequence of long-distance run

Blanka Hauptmannová [a,*], Jiří Votruba [b], Petr Neužil [c], Štěpán Černý [c], Miroslava Benešová [c], František Kölbel [d]

[a] Canadian Medical Care, Prague, Czech Republic
[b] Pulmonary Ward, VFN and 1.LFUK, Prague, Czech Republic
[c] Na Homolce Hospital, Prague, Czech Republic
[d] 2.LFUK and FN Motol, Prague, Czech Republic

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ABSTRACT

The acute mitral insufficiency is a life-threatening condition that may be caused by heavy physical strain, especially during the simultaneous occurrence of the myxomatous degeneration of the mitral valve. The mortality of the untreated illness is 75% during the first 24 h after the occurrence; the perioperative mortality is also high.

The case study describes the story of a 57-year-old male, an active sportsman (long-distance runner), whose health condition was duly examined in an institute of sport medicine in Germany, and the results were reportedly always normal. Immediately after finishing a marathon run here, in Prague, he began to complain of severe dyspnea, NYHA III–IV. The physical examination revealed clearly audible strong systolic murmur with the amplitude on the heart apex with propagation into the left axilla, and the signs of pulmonary congestion were present. The transthoracic echocardiography confirmed the suspected acute mitral regurgitation, with a minor dilatation of the left atrium, and the hyperkinetic left ventricle with the preserved systolic function (EF 70%).

After the confirming examination using transoesophageal echocardiography, the surgical revision of the mitral valve was indicated. This revealed myxoid degeneration of both leaflets of the mitral valve, tendinous cord rupture, and the dilatation of its annulus. A successful preservation operation was performed, together with the mitral plastic surgery, annuloplasty, and tendinous cord replacement. Having recovered from the difficulties of the post-surgery period, the patient was released to the domestic care on the 14th day after the operation.

* *Corresponding author at*: Canadian Medical Care, Veleslavínská 1, 162 02 Praha 6, Czech Republic. Tel.: +420 235 360 133.
E-mail address: blanka.hauptmannova@gmail.com (B. Hauptmannová).

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Introduction

The mitral valve is a very complicated structure, consisting of many parts and elements. Its correct function depends entirely on the complex functionality of all elements of the valvular apparatus – i.e. mitral annulus, both leaflets of the valve, tendinous cords, papillary muscles, but also the myocardium of the left atrium and the left ventricle [1]. The correct function therefore depends on the correct functionality and coordination of all the parts, and the well-preserved geometry of the whole complex. An acute damage of any part may result in the life-threatening condition – acute mitral insufficiency. The trigger may also be heavy physical strain, such as the long-distance run.

The most frequent cause of the acute mitral insufficiency is the damage of the papillary muscle caused by ischemia, rupture of the fixation apparatus, or the perforation of the valve leaflets during the infectious endocarditis, or upon the myxomatous degeneration or fibroelastic deficiency, mostly affecting elderly patients [2].

The clinical image presents, above all, the symptoms of the severe lung congestion with the signs of the left-sided heart failure, which may result in a severe shock. The treatment of patients suffering from a*cute mitral regurgitation involves the use of* vasodilating drugs, and both pharmaceutical and mechanical support of the cardiovascular system, but the only life-saving method is immediate surgery.

The mortality of the untreated illness varies according to the cause of occurrence, scope of the valve damage, and also according to the function of the left ventricle and the findings on coronary arteries. The mortality can be high; in case of a severe damage to the valve and the bad condition of the left ventricle, it can reach up to 50% within the first 24 h, and up to 90% within 48 h following the occurrence. The perioperative mortality is also high (20–50%) [3].

Observation

Our workplace (Canadian Medical Care, Praha, CMC) was examined and treated a 57-year old male – a foreigner, active long-distance runner, who was regularly examined at a clinic of sport medicine in Germany, allegedly always with negative results. The CMC examined him due to major dyspnea (NYHA III–IV) that occurred after he had finished the marathon run.

The initial physical examination showed, above all, clearly audible strong holosystolic murmur above the mitral valve, with the amplitude on the heart apex (5/6) with propagation into the axilla. Also present were symptoms of pulmonary congestion and tachycardia 126/min, blood pressure 130/90. The ECG recorded the sinus rhythm with the borderline AV conduction, normal range of QRS complex and length of QT interval, and the elevation of ST segment in the leads V5, V6. The resting O_2 saturation was 88%.

Based on the history and physical examination, the patient was immediately sent for further examination to the cardiology ward of Hospital Na Homolce, with suspected acute mitral insufficiency.

At the ward, the transthoracic echocardiography confirmed the suspected acute mitral regurgitation, with a minor dilatation of the left atrium, and the hyperkinetic left ventricle with the fully preserved function of the left ventricle (EF LK 70%).

The subsequent transoesophageal echocardiography confirmed the occurrence of major mitral regurgitation with the myxomatous degeneration of the rear valve leaflet, P2-Barlow prolapse, and the ''flail leaflet''.

The chest X-ray describes the increased lung congestion on the X-ray located paracardially on the right, or almost diffusely in left lung. The cardiac shadow is extended to the left.

Excerpts from the available lab results: Troponin I 0.14 ng/l, Myoglobin 47.4 ng/l, CRP 51.90 mg/l, leukocytes 12.9 g/l, D-dimers 168.0 ng/ml.

The selective coronarography showed normal findings on coronary arteries, dilated left ventricle with good wall kinetics, and severe mitral regurgitation of the 4th degree.

The patient was stabilized using the intra-aortic balloon counterpulsation (IABK) with the optimized after load, and indicated for the emergency cardiac surgery.

Again, the perioperative transoesophageal echocardiography gave yet another evidence of the major mitral regurgitation of the 4th degree, with a broad jet to the whole left atrium, good systolic functionality and kinetics of the left ventricle, myxomatous degeneration of the rear valve leaflet with the P2 prolapse, and the ''flail leaflet''. The aortal and tricuspid valve does not show any signs of regurgitation, the findings were normal.

During the surgery, the revision of the mitral valve confirmed the diagnosis of myxomatous changes of both leaflets (morbus Barlow), ruptured tendinous cords to the whole P2 segment, and massive dilatation of the annulus. The plastic surgery of the mitral valve was performed with a partial resection, transposition and re-implantation of the rear leaflet of the mitral valve, together with the tendinous cord replacement, and annuloplasty.

In the early post-operational stage, the patient was stable in terms of both hemodynamics and ventilation; gradually, inotropic support and IABK were discontinued.

Further post-operative development was complicated by the paroxysmal atrial fibrillation with spontaneous termination, and the AV block of 2nd degree Wenckebach (the borderline AV conduction was present even before the hospitalization). Given the missing symptomatology, and upon the consultation with the patient, the implantation of the pacemaker was rejected; further decisions will be based on the findings from the examinations at the patient's residency. The patient was released to the domestic care on the 14th day after the operation.

Discussion

Biochemically, the myxomatous degeneration of the mitral valve is characterized by the thickening and proliferation of the spongiform layer of the valve, with accumulation of glycosaminoglycans. This damages the firm fibrous structure of the valve, resulting in the presence of cystic structures and less dense collagen [4]. The changes in

collagen are indicated by the fragmentation of collagen fibers in the fibrosis, the elastic fibers are fragmented and form amorphous clusters. A typical example is the Barlow disease. The mitral leaflets are of myxoid appearance, the mitral ring is dilated, and the tendinous cords may be thickened or thin, are elongated, and often fragile, which may result in ruptures.

The functional consequence is that the affected leaflet of the valve prolapses into the left atrium during the systole, while the loose ends of both valve leaflets remained in touch below the level of the mitral annulus. Very often there is a combination of the myxomatous change of the valve and the rupture of the tendinous cord. All these changes are present for a long time, sometimes since the early childhood, and these patients can show no clinical symptoms at all.

In our case, the hemodynamic catastrophe of the modified valve only occurred after the extreme strain during the marathon run. Of course, we may speculate why the valve was not damaged during a routine training run. Certainly, the stress and excessive emotional strain during the race played its part – the excessive strain of the valve occurred, resulting in the increased contractility and rupture of the rear leaflet of the valve.

Sport activities, apart from their positive aspects, therefore have negative consequences as well – they increase the risk of sudden death, as the strain and mental stress increase the risk of sudden cardiac arrest for people with hidden cardiac problems.

There are various causes of death during sport activities. For people below 35 years, the most frequent are congenital disorders of the heart muscle (hypertrophic cardiomyopathy, abnormalities of the coronary arteries, arrhythmogenic cardiomyopathy of the right ventricle, heart rhythm disorders); for sportspeople over 35 years, it is ischemic heart disease.

As the preventive examinations of sportspeople do not include standard echocardiography, there is no chance to discover the signs of the myxoid degeneration of the mitral valve, which explains the normal findings during the preventive examinations. On the other hand, it is very hard to rule out suspected borderline findings outside the top performance. Therefore, it is only the races where the unexpected acute, and often nearly fatal, cardiac attack occurs, as described by our case study.

The literature cites several studies of death during sport activities; however, most only describe the cases of top sportspeople below 35 years.

The first thorough study that included the older population was that of Thomson (JAMA, 1982), investigating the incidence of deaths whilst jogging at Rhode Island between 1975 and 1980, in male population between 30 and 64 years of age [5]. The results show that there were 12 deaths in total, 11 of them caused by the ischemic heart disease. The authors found that the incidence of death among 30–64 year olds in runners, particularly men, is 1 case per 7620 runners. Another study investigating the deaths of amateur sportspeople was conducted in Ireland between 1987 and 1996; during these years, 51 cases were reported, with the average age of 48 years. 50 deceased were men, the most frequent sport during which the death occurred was golf, and in 42 cases, the cause of death was the ischemic heart disease [6].

The prevention of sudden deaths (SD) of sportspeople is still a hot topic. We must regard the fact that among top sports people, the risk of SD is 2.5 times higher than among the standard population. Among men, the risk is far higher than among women (9:1). This may be explained by the fact that, at least until recently, most men do sports that require higher physical activity.

In the sport medicine, the issue of full-body screening is still alive and important. What should such an examination include?

Nowadays, only Italy has established compulsory medical examinations before any race. This decision was based on the Maron study (1996), investigating the deaths of 158 sportspeople resulting from complicated hypertrophic cardiomyopathy [7]. Another study that supported the compulsory medical examinations was conducted in Italy as well (Dr. Corrado, Padua, 2003). The authors investigated retrospectively all sudden deaths of people below 35 years in the Veneto Region between 1979 and 1999. The results showed 51 deaths at the age between 12 and 35 [6].

The screening program of professional sportspeople from the Veneto Region resulted in the decrease of SD of sportspeople in this region by the incredible 89% in 25 years. From the original number of 3.5 in 100,000 sportspeople per year, the death rate decreased to 0.4 in 100,000 sportspeople per year, i.e. less than among non-sporting young population (about 1.0 in 100,000 people per year) [8].

These inspections are based on detailed history, including family history, and resting ECG. 8–10% of detected pathological conditions are sent to further detailed examinations.

The recommendation of the American Cardiology Society – the ''Preparticipation Screening'' includes the family history aimed at premature death with cardiac-related causes in the family, disability with cardiac-related causes below 50 years of age, hypertrophic or dilated cardiomyopathy, Marfan syndrome, severe arrhythmias, and inexplicable sudden death at the young age. This is followed by personal anamnesis with special focus on pain in the chest during physical strain, discomfort, conditions of collapse or pre-collapse during the strain, previously detected murmur, or high blood pressure. During the physical examination, it is recommended to focus on murmurs, pulse in femoral arterias to rule out the coarctation of the aorta, signs of the Marfan syndrome, and hypertension.

The consensual suggestion of the European Sport Cardiology Group to perform the preparticipation screening of sportspeople below 35 years (2005) includes anamnesis, resting ECG and physical examination. This screening should start as early as the period of intensive sport strain, i.e. at the age of 12–14, and should be repeated every 2 years. In case of positive findings, further specialized examinations are recommended.

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Conflict of interest

No conflict of interest of any author.

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